

Flow redistribution in the major cerebral arteries after carotid endarterectomy: A study with transcranial Doppler scan

E. M. Vriens, MD, PhD,^a G. H. Wieneke, PhD,^a B. Hillen, MD, PhD,^b B. C. Eikelboom, MD, PhD,^c
A. C. Van Huffelen, MD, PhD,^a and G. H. Visser, MD, PhD,^d *Utrecht, The Netherlands*

Purpose: This open single-center prospective study aimed to determine the redistribution of blood flow within the circle of Willis and through collateral pathways after carotid endarterectomy. Blood flow velocity and flow direction in the major cerebral arteries were determined, both at rest and during CO₂ inhalation.

Methods: Carotid endarterectomy was performed in 148 patients with a 70% or greater diameter stenosis of the internal carotid artery while patients were under general anesthesia. Arteriotomy closure was done with a venous patch. Selective shunting was performed with an electroencephalogram. Baseline blood flow velocity of the basal cerebral arteries was measured by means of transcranial Doppler sonography preoperatively (within 1 week before surgery) and 3 months postoperatively. At the same times, cerebrovascular reactivity was calculated during CO₂ inhalation insonating both middle cerebral arteries.

Results: Baseline blood flow velocity in the ipsilateral middle cerebral artery hardly changed 3 months postoperatively, but there was a considerable redistribution of flow in the circle of Willis. This was characterized by a decrease in contribution from the contralateral hemisphere through the anterior communicating artery, reduced cerebropetal flow rates in the ophthalmic artery, and smaller contribution of the posterior collateral sources. The CO₂ reactivity on the side of surgery increased in all patients. In patients with a contralateral occlusion, CO₂ reactivity increased on both sides. The redistribution of flow was most pronounced in patients who needed intraoperative shunting and in patients with a contralateral internal carotid artery occlusion.

Conclusion: After carotid endarterectomy, flow redistribution, as expressed by changes in blood flow velocity values, occurs in the circle of Willis. The contribution of collateral sources is diminished, and the CO₂ reactivity increases, both of which reflect improvement of the hemodynamic condition. The most improvement occurs in patients with contralateral occlusion. (*J Vasc Surg* 2001;33:139-47.)

The aim of carotid endarterectomy (CEA) is to prevent stroke, and large multicenter trials have shown an overall beneficial effect of CEA in patients with a severe internal carotid artery (ICA) stenosis and a recent nondisabling carotid-territory ischemic event.^{1,2} Although the stenotic lesion has been considered a source of embolism for many years, there is a growing awareness that such lesions also have hemodynamic effects, the impact of which is determined by the severity of the stenosis and by the capacity of the collateral circulation.³ The circle of Willis provides for several collateral pathways. The anterior communicating artery (ACoA) plays an important role in the balance of flow between the two hemispheres, and the posterior communicating artery (PCoA) has a similar function between the anterior and the posterior circulation.⁴ Other collat-

eral pathways include the external carotid artery (ECA), by reversed flow in the ophthalmic artery (OA), and leptomeningeal anastomoses. When the collateral circulation becomes inadequate and cerebral perfusion pressure decreases, there is compensatory vasodilatation of the arteriolar network, indicated by means of a lowered CO₂ reactivity.⁵ Both the degree of carotid artery stenosis and the ACoA diameter determine whether the perfusion pressure is adequate to maintain autoregulation.

Several studies have shown that CEA improves cerebral circulation⁶⁻⁸ and brain function^{9,10} after CEA. Improvement of cerebrovascular reactivity has been found with both CBF^{11,12} and transcranial Doppler (TCD) measurements.¹³⁻¹⁵ Moreover, health-related quality of life is significantly improved in patients with an occlusion of the contralateral ICA.¹⁶ TCD sonography permits quantitative investigation of blood flow velocity (BFV) and flow direction in the major cerebral arteries and the identification of several intracerebral collateral patterns.¹⁷ Using TCD recordings of the middle cerebral artery (MCA), some investigators found that BFV was increased after CEA,¹⁸⁻¹⁹ whereas most other investigators found no differences.²⁰⁻²² Moreover, there is only information on the redistribution of blood flow in the circle of Willis after CEA with respect to the anterior circulation.²¹

The aim of the current study was to study changes in BFV and flow direction in the circle of Willis and the oph-

From the Department of Clinical Neurophysiology,^a the Department of Functional Anatomy,^b the Department of Vascular Surgery,^c University Medical Centre Utrecht and the Rudolf Magnus Institute for Neurosciences; and the Department of Clinical Neurophysiology, Erasmus University Rotterdam.^d

Competition of interest: nil.

Reprint requests: A. C. Van Huffelen, Department of Clinical Neurophysiology (F 02.230), University Medical Center Utrecht, PO Box 85 500, 3508 GA Utrecht, The Netherlands (e-mail: a.c.vanhuffelen@neuro.azu.nl).

Copyright © 2001 by The Society for Vascular Surgery and The American Association for Vascular Surgery

0741-5214/2001/\$35.00 + 0 24/1/109768

doi:10.1067/mva.2001.109768

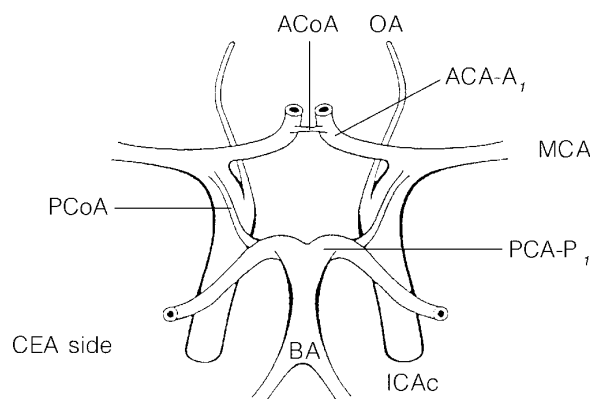


Fig 1. Circle of Willis. Data from the side of surgery are reported on the *left side*; data from the contralateral side are presented on the *right side*. ICA, Internal carotid artery; BA, basilar artery; MCA, middle cerebral artery; ACA-A₁, anterior cerebral artery; PCA-P₁, posterior cerebral artery; OA, ophthalmic artery; ACoA, anterior communicating artery; PCoA, posterior communicating artery; CEA, carotid endarterectomy.

thamic arteries as well as CO₂ reactivity at the arteriolar level in the territory of the MCA 3 months after CEA. We expected that by then flow redistribution through collateral pathways and a new equilibrium of vasodilatation would have been attained. We additionally studied whether the presence of different degrees of stenosis or occlusion of the contralateral ICA would lead to different changes after CEA.

METHODS

Study population

In a consecutive series of 150 patients, CEA was performed for an ICA stenosis $\geq 70\%$. In all patients, preoperative and postoperative TCD examinations were obtained. Patients were included from October 1994 until August 1996. Two patients were excluded because the ICA operated on was not patent during postoperative echo-duplex control, resulting in a study population of 148 patients. The ICA stenosis was examined with angiography or duplex technique. The criterion for a stenosis was $\geq 70\%$ according to the North American Symptomatic Carotid Endarterectomy Trial criteria or a peak systolic velocity 210 cm/s or greater. ICA stenoses $\geq 70\%$ were revealed in 6.1% of the patients by means of postoperative duplex investigations. The Institutional Review Board of the University Hospital Utrecht approved the study, and the patients gave their informed consent.

Patient characteristics

The median age of the patients was 66 years (5th, 95th percentile = 50, 80 years). Most patients were men (108 [73%]). The patients were classified according to the presence of permanent cerebral signs (nondisabling stroke), transient symptoms, or the absence of cerebral symptoms (Table I). The severity of ICA stenosis or occlusion on the

Table I. Patient characteristics

	N	%
Overall	148	100
Age (median, 5th and 95th percentile), y	66	50-80
Sex		
Men	108	73
Women	40	27
CEA-side		
Asymptomatic	49	33.1
Ocular	24	16.2
TIA	55	37.2
Minor stroke	20	13.5
Contralateral side		
Stenosis < 70%	86	57.8
Stenosis 70%-99%	30	20.4
Occlusion	32	21.8
Intraoperative EEG changes		
Shunt	24	16.2
No shunt	124	83.8

contralateral side was also recorded. In the year before surgery, 20 patients (13.5%) sustained a nondisabling stroke on the CEA-side hemisphere, and 55 patients (37.2%) had a CEA-side transient ischemic attack (TIA). Twenty-four patients (16.2%) had experienced only CEA-side ocular symptoms (transient monocular blindness or chronic retinal ischemia). Thirty-one patients (20.9%) were asymptomatic, and 18 patients (12.2%) had experienced symptoms on the contralateral hemisphere. The strictly asymptomatic patients were included in the Asymptomatic Carotid Surgery Trial. Moreover, in our hospital, CEA is performed to an asymptomatic > 70% stenosed ICA contralateral to an occluded ICA. In 32 patients, the contralateral ICA was occluded (21.8%), in 30 patients (20.4%) a high-grade contralateral stenosis (70%-99%) was present, and 86 subjects (57.8%) had < 70% contralateral ICA stenosis.

Intraoperative electroencephalographic monitoring

For the prevention of intraoperative cerebral ischemia, shunting was performed in selected patients, who were identified by means of electroencephalographic (EEG) monitoring. The criterion for the use of a shunt was the occurrence of unilateral and/or bilateral EEG slowing or a decrease of fast activity after carotid cross clamping. In 24 patients (16.2%), an endovascular shunt (Javid) was used. In most patients, a venous patch closed the arteriotomy.

Complications

After surgery, five patients had neurologic deficit. In two patients, a minor deficit remained, and in three patients, the symptoms resolved within 24 hours. In the first postoperative week, a nondisabling stroke (Rankin 3) developed in one patient, and two patients had a TIA. In the follow-up period of 3 months, an additional nondisabling stroke occurred, one patient had ocular symptoms, and three patients had a TIA (Table I). These patients were not excluded from the analysis.

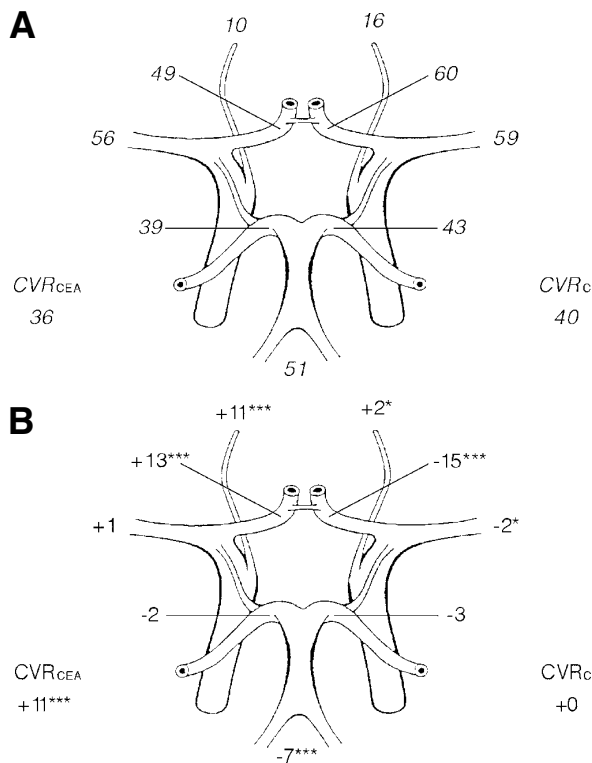


Fig 2. Blood flow velocity (cm/s) for different arteries and CO_2 reactivity (CVR, [%]) for the total group. **A**, Preoperative values and **B**, changes after CEA are shown. * $P < .05$, *** $P < .001$, all with Wilcoxon matched-pairs signed-ranks test.

TCD scan

TCD examinations were performed preoperatively and 3 months after surgery, by the use of a DWL Multidop-X device with two 2-MHz pulsed Doppler probes. (The OA was insonated with a 4-MHz probe). We used the time-averaged mean of the maximum BFV values as the TCD variable. The maximum BFV represents the maximum velocity (or Doppler frequency shift) measured at any moment of the cardiac cycle.

Baseline TCD scan. The investigation included bilateral insonation of the MCA, the A_1 segment of the anterior cerebral artery (ACA), the P_1 segment of the posterior cerebral artery (PCA), the OA, and the basilar artery (Fig 1). Special attention was paid to the direction of blood flow in the ACAs, which is indicative of collateral flow through the ACoA, and to the direction of blood flow in the OA, which in cases of flow reversal points to a decreased intra-arterial pressure in the corresponding distal part of the ICA. The side of the operation is referred to as the *CEA-side*, and the other side is referred to as the *contralateral side*, to avoid confusion about references to side. When an intracranial vessel was not found, this was treated as a missing value and was not included in the statistical analysis.

CO_2 reactivity. Immediately after the baseline investigation, the CO_2 reactivity for each hemisphere was deter-

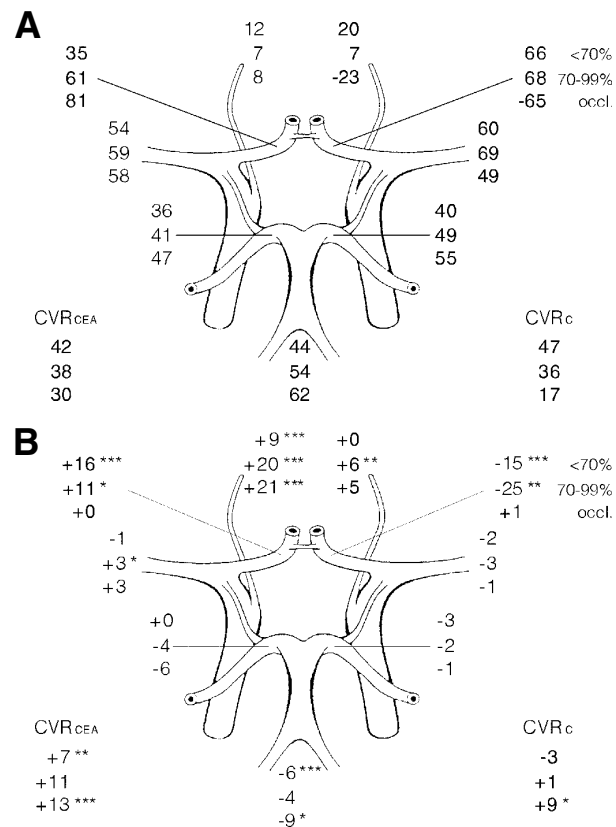


Fig 3. Blood flow velocity (cm/s) for different arteries and CO_2 reactivity (CVR, [%]) for three groups on the basis of contralateral stenosis. The first group is patients without hemodynamic contralateral ICA stenosis. The patients in the second group have 70% to 90% contralateral ICA stenosis. In patients in the last group, the contralateral ICA is occluded. **A**, Preoperative values and **B**, changes after CEA are shown. * $P < .05$, ** $P < .01$, *** $P < .001$, all with Wilcoxon matched-pairs signed-ranks test. Significant difference between the groups is printed in **bold** (Kruskal-Wallis). CVR_C , CO_2 reactivity at the contralateral side; CVR_{CEA} , CO_2 reactivity at the side of surgery.

mined by simultaneously insonating both MCAs, with the patient in a supine position with the eyes closed. The TCD probes were fitted into a metal frame fixed to the head with two earpieces and an adjustable nose saddle. A gas mixture of 5% CO_2 and 95% oxygen was inhaled through a mouthpiece connected to a respiratory balloon, and the CO_2 content of the breathed air/gas was continuously monitored with an infrared gas analyzer. After a resting period, the CO_2 reactivity was calculated from BFV at baseline and the BFV obtained after 1.5 minutes of inhalation of the gas mixture, and it was expressed as a relative (%) change.

Analysis

BFV values are expressed in centimeters per seconds. The median value and the 5th and 95th percentiles are reported. Distribution of several of the BFV values was skewed and contained extreme values; therefore, nonpara-

Table II. Blood flow velocity (cm/s) and CVR (%) for the total group

Artery	N	Preoperative values		Changes after CEA			Normal values	
		Median	Range	Median	%	Range	[x] (± 2 SD)	
CEA-side								
OA	126	10	−46, 27	+11*	+110%	−10, 62	24	8, 40
ACA-A ₁	103	49	−101, 113	+13*	+27%	−40, 166	50	26, 74
MCA	140	56	32, 92	+1	+2%	−20, 32	62	38, 68
PCA-P ₁	100	39	21, 81	−2	−5%	−49, 25	42	22, 62
BA	98	51	25, 92	−7*	−14%	−38, 16	42	22, 62
CVR	120	36	4, 66	+11*	+31%	−29, 47		
Contralateral side								
OA	132	16	−43, 37	+2†	+13%	−15, 21	24	8, 40
ACA-A ₁	101	60	−97, 116	−15*	−25%	−108, 31	50	26, 74
MCA	142	59	35, 88	−2†	−3%	−21, 24	62	38, 68
PCA-P ₁	92	43	20, 121	−3	−7%	−32, 25	42	22, 62
CVR	116	40	0, 75	0	0%	−35, 29		

For the various arteries, sample sizes differ, because different TCD data are missing in each case when the relevant artery was not found.

* $P < .001$, Wilcoxon matched-pairs signed-ranks test.

† $P < .05$, Wilcoxon matched-pairs signed-ranks test.

N, number of cases; range, 5th and 95th percentile; normal values, mean ± 2 SD (cm/s), adapted from literature.^{26,27}

metric statistical tests were used. Differences between preoperative and postoperative measurements were computed with the Wilcoxon matched-pairs signed-ranks test. Missing values were excluded pairwise. Differences between groups were calculated with the Kruskal-Wallis or Mann-Whitney test. P values less than .05 were reported.

RESULTS

Dropouts

Some of the ultrasound scanning measurements could not be performed because the ultrasound scan window in the temporal bone could not be found in six patients (4%), four times on the CEA-side, and two times on the contralateral side.

Total group

The preoperative median BFV and the changes in BFV after CEA were presented for the entire group of patients (Fig 2; Table II). The difference in preoperative median BFV values between mean normal and median value among patients is small.^{23,24} However, variation among patients is much larger than among control subjects because of highly abnormal values, especially in the ACA and OA, in which reversal of flow may be present.

After CEA, the BFV in both MCAs changed only slightly. In contrast, the BFV in the ACA_{CEA} increased considerably, with a corresponding decrease in the ACA_{contra}. This indicates that there was less collateral flow recruitment from the contralateral hemisphere. In addition, the BFV in the OA_{CEA} increased, (cerebrofugal or outward flow direction), as it did, but to a lesser extent in the OA_{contra}. This indicates that there was decreased recruitment of the collateral circulation through the ECA. Finally, the BFV in the BA decreased, which suggests that there was a decreased demand from the posterior circulation, although the BFV in both P₁ segments did not

change significantly. CO₂ reactivity on the CEA-side increased, but did not change on the contralateral side.

Stenosis of the contralateral internal carotid artery

The patients were divided into two groups: those with and those without contralateral ICA occlusion. The group without contralateral occlusion was subdivided into a group of patients with high-grade stenosis (70%-99%) and a group of patients with no or low-grade (< 70%) contralateral stenosis (Fig 3; Table III).

Contralateral ICA occlusion. Preoperatively, the group of patients with a contralateral ICA occlusion could be clearly distinguished from the other groups by a lower BFV in the MCA on the contralateral side, an increased BFV in the ACA_{CEA}, reversed flow in the ACA_{contra} and OA_{contra}, and a more pronounced contribution of flow in the posterior circulation (Fig 3). Moreover, the CO₂ reactivity on the CEA-side was at the lower limit and was clearly diminished on the ICA-occluded side.

After CEA, the hemodynamics of this group were improved, and this was evidenced by an increase in outwardly directed flow in the OA_{CEA}, (ie, a decrease of inwardly directed collateral flow), reduced flow rates in the posterior circulation (BA), and improvement of CO₂ reactivity on both sides. These changes indicate that collateral contribution from other sources had decreased. However, flow through the anterior part of the circle of Willis did not change significantly, indicating that the contribution of flow from the CEA-side hemisphere through the ACoA to the contralateral side was still maximally needed in this group.

Contralateral stenosis. In the other two groups of patients, those with low-grade or high-grade significant contralateral stenosis, BFV increased in the ACA_{CEA} and decreased in the ACA_{contra}, but to a different extent in the two groups. These changes reflect a redistribution of flow

Table III. Blood flow velocity (cm/s) for different arteries and CVR (%) for three groups of contralateral ICA stenosis

		Preoperative values		Changes after CEA		
	N	Median	Range	Median	%	Range
Ophthalmic artery						
CEA-side						
< 70%	75	12	-32, 29	+9*	+75%	-12, 50
70%-99%	24	7	-52, 25	+20*	+285%	-18, 81
Occlusion	26	8	-49, 30	+21*	+263%	-43, 68
P value		NS		NS		
Contralateral						
< 70%	81	20	8, 39	0	+0%	-10, 16
70%-99%	26	7	-33, 29	+6†	+86%	-7, 54
Occlusion	24	-23	-59, 41	+5	+22%	-80, 37
P value		.000		.018		
Anterior cerebral artery						
CEA-side						
< 70%	59	35	-104, 65	+16*	+46%	-17, 166
70%-99%	18	61	-138, 144	+11‡	+18%	-141, 262
Occlusion	25	81	40, 134	0	+0%	-165, 98
P value		.000		.003		
Contralateral						
< 70%	64	66	33, 134	-15*	-23%	-67, 23
70%-99%	18	68	-111, 160	-25†	-37%	-129, 19
Occlusion	19	-65	-132, 68	+1	+2%	-178, 222
P value		.000		NS		
Middle cerebral artery						
CEA-side						
< 70%	81	54	26, 84	-1	-2%	-19, 36
70%-99%	28	59	38, 100	+3‡	+5%	-9, 31
Occlusion	30	58	28, 95	+3	+5%	-30, 38
P value		NS		NS		
Contralateral						
< 70%	82	60	40, 112	-2	-3%	-24, 27
70%-99%	28	69	36, 100	-3	-4%	-28, 29
Occlusion	31	49	33, 77	-1	-2%	-24, 44
P value		.000		NS		
Posterior cerebral artery						
CEA-side						
< 70%	62	36	20, 80	0	+0%	-39, 23
70%-99%	21	41	19, 76	-4	-10%	-49, 109
Occlusion	23	47	24, 107	-6	-13%	-111, 26
P value		NS		NS		
Contralateral						
< 70%	55	40	20, 78	-3	-8%	-31, 20
70%-99%	19	49	21, 130	-2	-4%	-32, 25
Occlusion	17	55	29, 147	-1	-2%	-131, 180
P value		.004		NS		
Basilar artery						
CEA-side						
< 70%	57	44	23, 81	-6*	-14%	-22, 12
70%-99%	18	54	25, 104	-4	-7%	-41, 17
Occlusion	23	62	35, 92	-9‡	-15%	-56, 38
P value		.028				
Contralateral						
< 70%	—			—		
70%-99%	—			—		
Occlusion	—			—		
P value		—		—		
Cerebrovascular reactivity						
CEA-side						
< 70%	69	42	8, 67	+7†	+17%	-27, 46
70%-99%	23	38	-5, 92	+11	+29%	-55, 50
Occlusion	28	30	-7, 57	+13*	+43%	-21, 48
P value		.010		NS		
Contralateral						
< 70%	67	47	27, 77	-3	-6%	-35, 27
70%-99%	22	36	3, 95	+1	+3%	-40, 34
Occlusion	27	17	-10, 44	+9‡	+53%	-26, 36
P value		.000		.028		

For the various arteries, sample sizes differ, because different TCD data are missing in each case when the relevant artery was not found.

* $P < .001$, Wilcoxon matched-pairs signed-ranks test.

† $P < .01$, Wilcoxon matched-pairs signed-ranks test.

‡ $P < .05$, Wilcoxon matched-pairs signed-ranks test.

N, number of cases; range, 5th and 95th percentile; P value, Kruskal-Wallis P value among the three groups of contralateral ICA stenosis; NS, not significant.

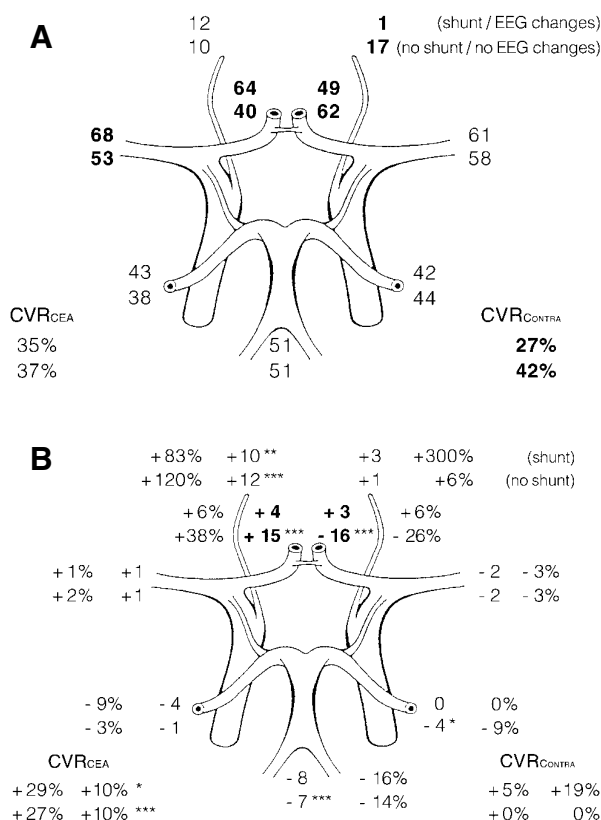


Fig 4. Blood flow velocity (cm/s) for different arteries and CO_2 reactivity (CVR, [%]) for two groups on the basis of shunt requirement. The first group consists of patients who needed a shunt because of intraoperative EEG changes. Patients in the second group did not need a shunt. **A**, Preoperative values and **B**, changes after CEA are shown. * $P < .05$, *** $P < .001$, all with Wilcoxon matched-pairs signed-ranks test. Significant difference between the groups is printed in **bold** (Mann-Whitney). CVR_{CEA} , CO_2 reactivity at the side of surgery; CVR_{CONTRA} , CO_2 reactivity at the contralateral side; EEG, electroencephalogram.

over the anterior part of the circle, with less suppletion from other sources. In these two groups, outward OA flow was also increased, especially on the CEA-side. The contribution of the basilar artery decreased in the low-grade stenosis group. The CO_2 reactivity increased on the side of surgery only in the low-grade stenosis group.

Need for intraoperative shunt insertion

In the preoperative condition, BFVs in the MCA and ACA on the CEA-side of shunted patients were higher than those in patients who did not need shunting, whereas the contralateral ophthalmic artery showed a significantly lower BFV (Fig 4; Table IV). In both groups, preoperative CO_2 reactivity (CVR) was within the reference range, but the contralateral CVR was lower in the group of patients who needed shunting.

After surgery, a significant difference was present between the two groups for the BFV in the ACAs. In the nonshunted group, a considerable amount of redistribution over the anterior part of the circle was present, expressed as an increase in BFV in the ipsilateral ACA and a decrease in the contralateral ACA, whereas this redistribution did not occur in the patients with a shunt.

Clinical classification

No significant differences were found between the different clinical subgroups (patients with nondisabling stroke, patients with TIA, patients with ocular symptoms, and asymptomatic patients). The pattern of redistribution after CEA did not differ from that found for the total group.

Blood pressure

Systolic and diastolic blood pressure and mean arterial blood pressure did not change significantly after the operation in the total group. In the between-group analysis, only the systolic blood pressure was significantly increased in the patients with a contralateral ICA occlusion.

DISCUSSION

We measured flow velocities in the major cerebral arteries 3 months after CEA to determine how carotid artery reconstruction affects the distribution of blood flow in the circle of Willis and in the OA. We chose this period because we expected that postoperative hemodynamic disturbances would have stabilized and that there would be as yet no new major overall atherosclerotic changes in the carotid arteries and intracranial vessels.

In many patients, the median preoperative BFV of the investigated efferent vessels was within the reference range. However, a large variance of data was found, because in some the patients' clearly abnormal BFVs were found. The abnormality was especially manifested through the reversal of flow in the ACA and OA in several patients. Postoperatively, the BFV in both MCAs was not significantly increased. There was a significant decrease in flow on the contralateral side. In contrast, there were pronounced changes in the BFV in the other vessels of the circle of Willis and in the OA, namely, a decrease in the collateral contribution from the contralateral side through the anterior communicating artery to the CEA-side (BFV- ACA_{CEA} increased, BFV- ACA_{CONTRA} decreased). The balance of flow through both the ACoA and the ipsilateral PCoA appeared to be "normalized." Collateral flow from the ECA through the OA was decreased, and there was a smaller contribution from the posterior collateral circulation. This decreased contribution of the collateral circulation can be explained by assuming that the flow territory of the MCA is the principal factor determining the collateral flow pattern. Normalization of the mean pressure reserve at the MCA entrance by reconstruction of the corresponding ICA diminishes the demand of the MCA for collateral supply. Although BFV in the MCA remained unchanged, there was a considerable increase in the CVR

Table IV. Shunt requirement: Blood flow velocity (cm/s) for different arteries and CVR (%) for patients with or without intraoperative EEG changes

		<i>Preoperative values</i>		<i>Changes after CEA</i>		
	<i>N</i>	<i>Median</i>	<i>Range</i>	<i>Median</i>	<i>%</i>	<i>Range</i>
Ophthalmic artery						
CEA-side						
Shunt	22	12	−78, 27	+10*	+83%	−10, 112
No shunt	104	10	−45, 30	+12†	+120%	−18, 59
<i>P</i> value		NS		NS		
Contralateral						
Shunt	22	1	−52, 33	+3	+300%	−15, 52
No shunt	118	17	−43, 39	+1	+6%	−18, 22
<i>P</i> value		.044		NS		
Anterior cerebral artery						
CEA-side						
Shunt	18	64	22, 134	+4	+6%	−172, 40
No shunt	94	40	−109, 109	+15†	+38%	−44, 168
<i>P</i> value		.001		.049		
Contralateral						
Shunt	16	49	−115, 144	+3	+6%	−145, 222
No shunt	102	62	−85, 115	−16†	−26%	−111, 34
<i>P</i> value		.026		.04		
Middle cerebral artery						
CEA-side						
Shunt	24	68	25, 130	+1	+1%	−34, 32
No shunt	117	53	32, 91	+1	+2%	−19, 33
<i>P</i> value		.011		NS		
Contralateral						
Shunt	23	61	27, 146	−2	−3%	−30, 46
No shunt	121	58	35, 85	−2	−3%	−18, 27
<i>P</i> value		NS		NS		
Posterior cerebral artery						
CEA-side						
Shunt	19	43	21, 100	−4	−9%	−56, 117
No shunt	90	38	20, 80	−1	−3%	−49, 26
<i>P</i> value		NS		NS		
Contralateral						
Shunt	16	42	17, 78	0	0%	−14, 32
No shunt	98	44	20, 122	−4‡	−9%	−38, 25
<i>P</i> value		NS		NS		
Basilar artery						
CEA-side						
Shunt	15	51	23, 92	−8	−16%	−60, 17
No shunt	89	51	25, 96	−7†	−14%	−38, 15
<i>P</i> value		NS		NS		
Contralateral						
Shunt	—			—		
No shunt	—			—		
<i>P</i> value		—		—		
Cerebrovascular reactivity						
CEA-side						
Shunt	20	35	−12, 84	+10	+29%	−35, 49
No shunt	115	37	4, 66	+10†	+27%	−29, 47
<i>P</i> value		NS		NS		
Contralateral						
Shunt	19	27	−8, 98	+5	+19%	−38, 25
No shunt	104	42	6, 75	0	0%	−35, 30
<i>P</i> value		.010		NS		

For the various arteries, sample sizes differ, because different TCD data are missing in each case when the relevant artery was not found.

**P* < .01, Wilcoxon matched-pairs signed-ranks test.

†*P* < .001, Wilcoxon matched-pairs signed-ranks test.

‡*P* < .05, Wilcoxon matched-pairs signed-ranks test.

N, number of cases; *range*, 5th and 95th percentile; *P* value, Mann-Whitney *P* value between the two groups of shunt requirement.

on the side of surgery in all patients. In patients with a contralateral occlusion, the CVR increased not only on the side of surgery, but also on the contralateral side. This is in agreement with previous findings of our group.¹⁵ This improvement of CVR reflects an increased cerebrovascular reserve capacity and, consequently, an improved hemodynamic condition.

In the group of patients with a contralateral occlusion, the flow velocity distribution over the anterior part of the circle after CEA is still the same, whereas collateral supply by the OA and posterior circulation had diminished. So, after surgery, the perfusion of the contralateral hemisphere was still provided by the CEA-side hemisphere through the ACoA.

After CEA, the flow velocity distribution over the anterior part of the circle hardly changed in patients requiring a shunt during surgery, whereas flow velocity over the ACoA was normalized in patients without a shunt. This may suggest that in patients requiring a shunt there is no or limited connection through the ACoA. Consequently, no blood supply is possible from the contralateral side during surgery, and no change in flow in the ACAs will occur after surgery. The finding of a statistically significant change in BFV in an artery does not mean that this change will occur in most or all patients or in a subset thereof.

There is little information in the literature about BFV after CEA, and what does exist is contradictory. Most attention has been focused on data for the MCA. Some authors reported no significant changes in BFV in the MCA after a period varying from 6 weeks to 3 months,^{20-22,25} whereas other authors detected a persistent increase in BFV in the MCA, although it was less pronounced than in the direct postoperative phase.^{18,19} Araki et al¹⁹ described a persistent increase in BFV in the MCA_{CEA} and in the ACA_{CEA}, but a decrease in BFV in the ACA_{contra} detected early after the operation was no longer detected after 3 months. They did not provide information on the posterior part of the circle of Willis, and they measured flow in the OA by means of oculoplethysmography rather than with TCD.

TCD sonography has some limitations. First, flow velocity is measured, not volume flow. The use of flow velocity as a means of inferring changes in flow volume is only justifiable when the diameter of the insonated vessel does not change. The constancy of diameter has been confirmed for two conditions, changes in CO₂^{26,27} and intraoperative changes during CEA.^{28,29} When BFV differences between the preoperative and postoperative condition are calculated, slow adaptation of vessel diameter after flow redistribution cannot be completely excluded. Second, the ACoA and two PCoA segments of the circle of Willis, which play an important role in flow redistribution, cannot be studied directly. Consequently, collateral blood flow through these arteries has to be derived indirectly. Presently, transcranial color-coded sonography is used as a means of obtaining more detailed information of the interrelation between flow pattern and vessel anatomy. Third, the interpretation of the intraoperative and early postoperative TCD scan recordings, relative to preoperative values, is hampered

by several confounding factors, such as anesthetic agents and hemodilution. The effect of these factors will gradually disappear with time, and a new equilibrium in the collateral circulation and cerebrovascular reactivity will be established. BFV is reported to be increased by 20% to 30% in the MCA in the early postoperative phase and by slightly less in the ACA and OA.¹⁷ However, as mentioned, these early changes are not always found at later times.^{18-20,25,30,31}

We consider the redistribution of flow over the circle of Willis and the increase in CO₂ reactivity to be two separate steps in the process of improvement of the hemodynamic condition after CEA. The first step takes place at the level of the large vessels at the base of the brain. The second step takes place at the level of the arterioles. In most groups of patients, however, both steps can be recognized, each contributing to a different extent.

To what extent the beneficial effects of CEA depend on the prevention of thromboembolism or on its hemodynamic consequences is not known. The results of this study indicate that, in at least some patients, improvement of the hemodynamic condition is an important result of CEA. On the basis of our results, we conclude that patients with contralateral occlusion would probably benefit most from surgery. Whether this beneficial hemodynamic effect also leads to a better clinical prognosis has to be studied in clinical trials. It is essential to stratify patients participating in such trials according to hemodynamic factors, such as the presence of contralateral ICA occlusion.

REFERENCES

1. European Carotid Surgery Trialists' Collaborative Group. Randomised trial of endarterectomy for recently symptomatic carotid stenosis: final results of the MRC European Carotid Surgery Trial (ECST). *Lancet* 1998;351:1379-87.
2. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. *N Engl J Med* 1998;339:1415-25.
3. Klijn CJM, Kappelle LJ, Tulleken CAF, Van Gijn J. Symptomatic carotid artery occlusion: a reappraisal of hemodynamic factors. *Stroke* 1997;28:2084-93.
4. Cassot F, Vergueur V, Bossuet P, Hillen B, Zagzoule M, Marc-Vergnes J-P. Effects of anterior communicating artery diameter on cerebral hemodynamics in internal carotid artery disease: a model study. *Circulation* 1995;92:3122-31.
5. Powers WJ. Cerebral hemodynamics in ischemic cerebrovascular disease. *Ann Neurol* 1991;29:231-40.
6. D'Addato M, Pedrini L, Stella A, Pecchi M, Monetti N, Dondi M, et al. Carotid endarterectomy: pre- and post-operative monitoring with cerebral SPECT. *Int Angiol* 1988;7:234-7.
7. Balm R, Van der Grond J, Mali WPTHM, Eikelboom BC. Reestablishment of cerebral metabolism after carotid endarterectomy. *Eur J Vasc Endovasc Surg* 1995;10:182-6.
8. Blankensteijn JD, Van der Grond J, Mali WPTHM, Eikelboom BC. Flow volume changes in the major cerebral arteries before and after carotid endarterectomy: an MR angiography study. *Eur J Vasc Endovasc Surg* 1997;14:446-50.
9. Irvine CD, Gardner FV, Davies AH, Lamont PM. Cognitive testing in patients undergoing carotid endarterectomy. *Eur J Vasc Endovasc Surg* 1998;15:195-204.
10. Lunn S, Crawley F, Harrison MJG, Brown MM, Newman SP. Impact of carotid endarterectomy upon cognitive functioning. *Cerebrovasc Dis* 1999;9:74-81.
11. Schroeder TV, Sillesen H, Engell HC. Hemodynamic effect of carotid endarterectomy. *Stroke* 1987;18:204-9.

12. Russell D, Dybevoeld S, Kjartansson O, Nyberg-Hansen R, Rootwelt K, Wiberg J. Cerebral vasoreactivity and blood flow before and 3 months after carotid endarterectomy. *Stroke* 1990;21:1029-32.
13. Markus HS, Harrison MJ, Adiseshiah M. Carotid endarterectomy improves haemodynamics on the contralateral side: implications for operating contralateral to an occluded carotid artery. *Br J Surg* 1993;80:170-2.
14. Bornstein NM, Gur AY, Shifrin EG, Morag BA. Does carotid endarterectomy modify cerebral vasomotor reactivity? *Cerebrovasc Dis* 1997;7:201-4.
15. Visser GH, Van Huffelen AC, Wieneke GH, Eikelboom BC. Bilateral increase in CO₂ reactivity after unilateral carotid endarterectomy. *Stroke* 1997;28:899-905.
16. Vriens EM, Post MWM, Jacobs HM, Van Huffelen AC, Eikelboom BC. Changes in health-related quality of life after carotid endarterectomy. *Eur J Vasc Endovasc Surg* 1998;16:395-400.
17. Schneider PA, Rossman ME, Torem S, Otis SM, Dilley RB, Bernstein EF. Transcranial Doppler in the management of extracranial cerebrovascular disease: implications in diagnosis and monitoring. *J Vasc Surg* 1988;7:223-31.
18. Blohmé L, Pagani M, Parra-Hoyos H, Olofsson P, Takolander R, Swedenborg J. Changes in middle cerebral artery flow velocity and pulsatility index after carotid endarterectomy. *Eur J Vasc Surg* 1991;5:659-63.
19. Araki CT, Babikian VL, Cantelmo NL, Johnson WC. Cerebrovascular hemodynamic changes associated with carotid endarterectomy. *J Vasc Surg* 1991;13:854-60.
20. Magee TR, Davies AH, Baird RN, Horrocks M. Transcranial Doppler measurement before and after carotid endarterectomy. *J R Coll Surg Edinb* 1992;37:311-2.
21. Van Huffelen AC, Vriens EM, Wieneke GH, Touw-Otten F, Eikelboom BC. Carotid endarterectomy: a three months follow-up study. *Electroenceph clin Neurophysiol* 1993;87:S52-3.
22. Manara R, Baracchini C, Ermani M, Meneghetti G. Intracranial hemodynamics pre-post carotid surgery. *Eur J Ultrasound* 1997;5:S10.
23. Adams RJ, Nichols FT, Hess DC. Normal values and physiological variables. In: Newell DW, Aaslid R, editors. *Transcranial Doppler*. New York: Raven Press; 1992. p. 41-8.
24. Transcranial Doppler ultrasonography: normal values. In: DeWitt LD, Rosengart A, Teal PA, Babikian VL, Wechsler LR, editors. *Transcranial Doppler ultrasonography*. St. Louis; 1990. p. 29-38.
25. Müller M, Schüder G, Langenscheidt G-P, Walter P, Schimrigk K. Perioperative TCD monitoring in carotid endarterectomy. *Eur J Ultrasound* 1997;5:S10-1.
26. Giller CA, Bowman G, Dyer H, Mootz L, Krippner W. Cerebral arterial diameters during changes in blood pressure and carbon dioxide during craniotomy. *Neurosurgery* 1993;32:737-42.
27. Valdueza JM, Balzer JO, Villringer A, Vogl TJ, Kutter R, Einhüpfel KM. Changes in blood flow velocity and diameter of the middle cerebral artery during hyperventilation: assessment with MR and transcranial Doppler sonography. *AJNR Am J Neuroradiol* 1998;18:1929-34.
28. Aaslid R, Lindegaard KF, Sorteberg W, Nornes H. Cerebral autoregulation dynamics in humans. *Stroke* 1989;20:45-52.
29. Lindegaard KF, Lundar T, Wiberg J, Sjøberg D, Aaslid R, Nornes H. Variations in middle cerebral artery blood flow investigated with noninvasive transcranial blood velocity measurements. *Stroke* 1987;18:1025-30.
30. Gossetti B, Martinelli O, Guerricchio R, Irace L, Benedetti-Valentini F. Transcranial Doppler in 178 patients before, during, and after carotid endarterectomy. *J Neuroimaging* 1997;7:213-6.
31. Steiger HJ, Schäffler L, Boll J, Liechti S. Results of microsurgical carotid endarterectomy: a prospective study with Transcranial Doppler, and EEG monitoring, and elective shunting. *Acta Neurochir (Wien)* 1989; 100:31-8.

Submitted Jan 25, 2000; accepted Apr 11, 2000.